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LETTERS AND CORRESPONDENCE

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Immediate or Delayed Therapy With 2-CdA for Hairy Cell Leukemia in a Jehova's Witness?

To the Editor: Couban and Wilson recently reported a Jehova's Witness with pneumonia of unknown ethiology and severe anemia due to hairy cell leukemia, who had immediate therapy with standard-dose-2-chlorodeoxyadenosine (cladribine, 2-CdA), and subsequently due to persistent cytopenia granulocyte colony-stimulating factor and erythropoietin. From my experience with 2-CdA treatment of more than 100 patients with advanced hairy cell leukemia, I find it unlikely that the recovery from cytopenia was accelerated by the addition of the growth factors. Despite the successful outcome of the presented case, I strongly discourage the management, since it is associated with an unacceptable risk of fatal complications, mainly opportunistic infections [1].

In the reported case, the leukocytes started to rise on the fourth week from start of 2-CdA, and the hemoglobin around the sixth week. This is identical to what we found in patients with febrile complications following 2-CdA with no growth factors [1]. In fact, we have also documented that granulocyte–macrophage colony-stimulating factor (GM-CSF) did not accelerate neutrophil recovery when given following 2-CdA in patients with hairy cell leukemia and advanced cytopenia (GM-CSF group n = 12, matched control group n = 15) [2].

More importantly, it is well documented that 2-CdA gives an early decrease in both hemoglobin and neutrophil counts [1,3], in addition to the dramatic fall of T-cell counts [4]. The early fatality rate from 2-CdA in hairy cell leukemia is 3% [2,3], with a much greater risk in patients with unresolved infection of unknown ethiology at the start of treatment [1]. Such patients, as well as patients with severe anemia who refuse transfusions should not be treated immediately with standard-dose 2-CdA. The safest way would be to start with interferon-α (IFN-α), which gives much less of early immune suppression and deterioration of anemia and await recovery from infection and some improvement from cytopenia. 2-CdA should be given as definitive therapy later. Another alternative would be to give low-dose CdA, i.e., 2 mg/m^2 daily for 7 days [5], or 5 mg/m^2 daily for 3 days (Juliusson et al., unpublished observations). This schedule gives the same time to recovery from cytopenia, with less immunosuppression, and probably less deterioration of anemia.

GUNNAR JULIUSSON

Department of Hematology, University Hospital, Linköping, Sweden

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Photosensitivity and Thrombocytopenia Due to Amitriptyline

To the Editor: A 68-year-old woman, with no familial or personal history, presented with a pruritic erythematous rash over the face, neck, and both hands (Fig. 1). She had received daily doses of 60 mg amitriptyline and 2 mg flunitrazepam for depression for approximately 4 months. Laboratory data revealed a platelet count of 31 × 10^9/l (normal, 110–340 × 10^9/l) and a prothrombin time of 10.6 sec (normal, 12–16 sec). A partial thromboplastin time and Lee-White clotting time were normal. Other blood and urine hematochemical values were within the normal range. Bone marrow aspiration showed megakaryocytic hyperplasia with many young megakaryocytes. Treatments with 40 mg of prednisone daily and fluocinonid cream were started. Within 3 days, the platelets increased to 110 × 10^9/l and the skin rash disappeared.

Patch and photopatch (UVA 5 J/cm^2) testings with amitriptyline (10 and 30% petrolatum) and flunitrazepam (10 and 30% pet.) were performed using Finn Chambers® (Epitest, Ltd., Helsinki, Finland) on Scanpor® tape (Norgesplaster A/S, Oslo, Norway) and were all negative. Ten milligrams of amitriptyline was orally given without sun protection and after 2 days an itchy erythematous rash appeared on the face and thrombocytopenia developed.

Amitriptyline is a widely used tricyclic antidepressant [1]. It is known to produce various cutaneous side effects such as dermatitis herpetiformis-like eruption, hyperpigmentation in synergism with minocycline, eczematous exanthema, vasculitis, or skin blisters [2–4]. Although amitriptyline can also induce thrombocytopenia and thrombocytopenic purpura [5], pho-